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ABSTRACT

A state-of-information survey concerning health effects of indoor air pollutants in the categories of formaldehyde, volatile organics, radon, particulates, and combustion products has been prepared by the U.S. EPA Environmental Criteria and Assessment Office, RTP, N.C. Included in each category are human effects, animal exposure experiments, mutagenicity, and carcinogenicity information. In addition to those health implications associated with each category of pollutants, the problems of measuring exposure to the complex mixtures actually breathed by people in homes is addressed.

INTRODUCTION

Most individuals spend 80 to 90% of their time indoors (Dockery and Spengler 1981; Szalai 1972; NAS 1981). While great efforts have been made to clean up the outdoor air in recent years, assessment of the impact of pollutants that people are exposed to for the greater part of their lives indoors has only recently begun.

The information gathered by the nation's air pollution control community in the last 15 years concerning such criteria pollutants as carbon monoxide (CO), particulate matter, nitrogen oxides (NO_x), especially nitrogen dioxide (NO_2), and other pollutants of potential hazard in the outdoor air such as formaldehyde (HCHO), polycyclic aromatic hydrocarbons (PAH), and volatile organic compounds (VOC) is a logical starting point in assessing their effect in indoor air. Measurement of individual concentrations and their health effects outdoors and in industrial settings, and from experimental studies, gives important indications of the impact to be expected from them indoors. It is important to remember, however, that what people actually breathe is a mixture of these various pollutants.

This paper attempts to synthesize a picture of indoor air pollution encountered in residences in the United States based on current health intelligence. It must be recognized that there is a plethora of variables that must be accounted for in the description of "residence." We summarize effects of individual components of indoor air pollution in tables that indicate measured indoor concentrations, where known, plus concentrations with their health effects.

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For the purpose of this preliminary discussion, it is useful to divide the complex mixture of indoor air pollutants into major categories whose effects have been studied and evaluated. The categories we have chosen are formaldehyde, combustion products (including carbon monoxide, nitrogen oxides, particulates, PAH), radon, and volatile organic compounds. Other categories that may be evaluated in a later paper are fibers, pesticides, dyes, and biological contaminants.

FORMALDEHYDE

Formaldehyde (HCHO) is a colorless gas at room temperatures. It has a pungent odor and is ubiquitously emitted from such sources as insulation, particleboard and plywood, and fabrics and carpets using aldehyde resins. It is also a product of incomplete combustion so that heating, cooking, and cigarette smoking contribute to the indoor burden. Indoor concentrations can be very high in prefabricated homes built with large amounts of chipboard construction materials. Many new homes use such materials in basic construction, as well as in furniture, cabinets, and carpeting and upholstery materials. Concentrations measured in prefabricated homes range from 0.1 to 5.0 mg/m^3, although the highest values occur rarely ($1\ mg/m^3 = 0.813$ ppm HCHO). More commonly the range is 0.1 to 1.0 mg/m^3. In conventionally built homes, recent surveys show concentrations averaging 0.05 to 0.1. Emission rates are influenced by temperature, humidity, ventilation rates, and the concentration in the air of the home.

Clinical and epidemiologic data indicate considerable variation in human response to formaldehyde, including hypersensitive reactions. Table 1 lists ranges and median responses to short-term exposure but does not include responses of immunosensitive populations.

Responses of animals to formaldehyde exposure show species variation. Animals typically show a reflex inhibition of respiration, produced when formaldehyde stimulates trigeminal nerve endings in the cornea and nasal mucosa (Chang et al. 1981). Mice are able to inhibit respiration more than rats or monkeys (Rusch et al. 1983a), which may minimize their exposure and account for fewer long-term lesions seen in mice vis-à-vis rats (Chang et al. 1981). Short-term exposures of animals result in signs of irritation of the eyes and nasal mucous membranes, such as lacrimation and increased mucus discharge and rhinitis (Rusch et al. 1983b).

A long-term study was carried out by researchers at the Chemical Industry Institute of Toxicology (CIIT) on Fischer 344 rats and $B_6C_3F_1$ mice. Groups of animals exposed for up to 24 months to concentrations of 14.3, 5.6, and 2.0 ppm (1 ppm formaldehyde = $1.25\ mg/m^3$) showed increasing numbers of increasingly severe lesions of the lining of the nasal cavity. The lesions included reversible squamous metaplasia, which was exposure-related in its severity, and squamous cell carcinomas in the high-exposure (14.3 ppm) group. Rats exposed to 5.6 ppm formaldehyde developed lesions seen in mice at the higher exposure level of 14.3 ppm (Kerns et al. 1983).

Rusch et al. (1983a) found squamous metaplasia in nasal turbinates of monkeys and in rats in a 3.0 ppm exposure group, as well as general signs of irritation. Hamsters exposed did not show tissue lesions. The animals in this study were exposed for 22 hours a day, seven days a week for 26 weeks. Total dose was higher than in the Kerns et al. (1983) study, but concentrations were less, indicating that concentrations of exposure may play a larger role than total dose.

Several recent epidemiologic studies have been conducted regarding the link between formaldehyde exposure and cancer (Olsen et al. 1984; Acheson et al. 1984; Hernberg et al. 1983; Olsen et al. in press; Blair et al. in press). All of these studies have weaknesses in the exposure data, since often exposures were not measured but only estimated. The power for detecting the rather rare nasopharyngeal cancer, which in humans is the type of cancer that had been found in rodents, was small. Controversy still exists regarding the carcinogenic role of formaldehyde in humans (Sun 1986).

The molecular/cellular mechanism of formaldehyde toxicity is not yet entirely clear. Evidence exists that formaldehyde is electrophilic for macromolecules, including DNA, RNA, and protein, and forms reversible adducts, as well as irreversible cross-linking (Feldman 1973; Casanova-Schmitz et al. 1984; Swenberg et al. 1983). Its specific role in promotion or initiation of carcinogenesis is still to be elucidated. Despite the somewhat equivocal and contradictory evidence from the various epidemiologic studies, the incidence of cancer in rodents makes formaldehyde highly suspect as a human carcinogen.

Irritant effects include increased cell membrane permeability, inflammation, and edema. Stutts et al. (1986) showed that formaldehyde inhibits surface membrane permeability to sodium and chloride ions in bronchial epithelial cells of dogs and postulated that this may affect mucociliary clearance. Anderson and Proctor (1982) showed decreased mucociliary clearance rates for human nasal epithelium exposed to formaldehyde. The effect of formaldehyde irritant effects on general respiratory health is not yet clear. Aside from chemical hypersensitivity, effects on the immune system and their sequelae have not been investigated.

COMBUSTION PRODUCTS

Combustion products are primarily released in the home by gas stoves, unvented kerosene heaters, woodstoves and fireplaces under downdraft conditions, and by cigarette smoking. While the composition of products released from incomplete combustion varies with the source and the conditions under which the source is operated, it can be categorized into groups that can be discussed individually.

Major categories of products from incomplete combustion can be listed as CO, NO_x, particulate material, and volatile and semivolatile polynuclear aromatic hydrocarbons (PAH).

CO is an odorless, colorless gas, which has about 250 times the affinity for hemoglobin that oxygen has. The formation of carboxyhemoglobin (COHb) reduces the amount of hemoglobin available for carrying oxygen to tissues, so that CO acts as an asphyxiating agent. Its low-level effects are summarized in Figure 1 and Table 2.

The term nitrogen oxides (NO_x) refers to such compounds of nitrogen as NO, NO_2, N_2O, OONO, ON(O)O, N_2O_3, N_2O_4, and N_2O_5, all of which are irritant gases that have the potential to affect human health. Of these, the effects of NO_2 have been most extensively studied. Animal studies have given evidence of the mechanisms of toxicity involved in NO_2 exposure, but for the most part animals have been exposed to much higher concentrations than humans encounter indoors (DOE 1985).

The most frequent and significant NO_2 respiratory effects seen at concentrations at or near ambient levels are

1. Altered lung function and symptomatic effects observed during controlled human exposure studies (Bauer 1984).

2. Increased incidence of acute respiration symptoms and illness seen in outdoor epidemiologic studies and indoor epidemiologic studies of homes using gas stoves (Melia et al. 1983; Melia et al. 1982a; Melia et al. 1982b; Ware et al. 1984).

3. Lung tissue damage and increased susceptibility to infection observed in animal toxicology studies (Kuraitis et al. 1981; McGrath and Oyervides 1983; McGrath and Smith 1984).

Table 3 indicates the effects of exposure to NO_2 (alone) for short time periods.

It is important to point out that controlled human exposure studies present conflicting and somewhat ambiguous results concerning respiratory effects of NO_2 in both asthmatics and healthy persons in concentrations in the range of 0.1 to 4.0 ppm NO_2 (1 ppm NO_2 = 1.9 mg/m^3). Some studies report increased airway resistance or decreased lung function when bronchoconstricting agents are administered along with NO_2 exposure (Ahmed et al. 1982; Kleinman et al. 1983; Bauer et al. 1984), while others (Hazucha et al. 1983; Ahmed et al. 1983) show no statistically significant effects. The relationship of NO_2 exposure to respiratory effects is also relatively weak in recent indoor epidemiologic studies done by Melia et al. (1982a,b); Melia et al. (1983); Speizer et al. (1980); Ware et al. (1984). Animal studies substantiate an effect on immune function from NO_2 exposure (Richters and Richters 1983; Richters and Kuraitas 1981; McGrath and Oyervides 1983; McGrath and Smith 1984; Lefkowitz et al. 1983; Lefkowitz et al. 1984), but the difficulty of extrapolating these findings from animals to humans remains.

Particulates are another combustion product that can affect lung function. The term represents a broad class both chemically and physically, found in the air in discrete particles, including liquid droplets. Size of particles determines the location and magnitude of impact of particles. Those particulates included in the respirable particle class

(<2.5 μm) present a risk to health greater than a comparable mass concentration of larger particles (<2.5 μm) since the smaller particles are breathed deep into the lungs, circumventing many respiratory defense mechanisms, and can deliver high concentrations of potentially harmful substances to a few cells that may be severely damaged.

Size also determines the surface area/total mass ratio, which increases with decrease in particle size. This becomes important in considering particle damage, since harmful chemical species, especially polynuclear aromatic hydrocarbons (PAH), adsorb onto the surface of particles, impact on cells, or can be taken up by cells, with the particle acting as carrier. Other particles may in themselves be chemically reactive (e.g., asbestos, acid) and damage cells of the lungs through chemical interaction.

Of concern in the health effects of respired particles are (1) chemical or mechanical irritation of tissues, including nerve endings at the site of deposition, (2) impairment of respiratory mechanics, (3) aggravation of existing respiratory or cardiovascular disease, (4) reduction in particle clearance and other host defense mechanisms, (5) impact on host immune system, (6) morphologic changes of lung tissues, (7) carcinogenesis.

Because combustion conditions affect the number, particle size, and chemical speciation of particles, and because these conditions vary rapidly, it becomes very difficult to predict specific health effects resulting from exposure to particulate matter expressed as an average mass concentration. Respirable particles at concentrations of 250 to 350 $\mu g/m^3$ increase respiratory symptoms in compromised individuals (U.S. EPA 1981, 1984). Longer-term effects depend largely on interaction with cells whose damage is harder to classify.

The effects of PAH from combustion sources are difficult to predict, not only because the chemical composition and concentration of these compounds vary with combustion conditions, but also because of the nature of the interaction of these compounds with human detoxifying systems. Some PAH are carcinogens; others are pro- or co-carcinogenic and may depend on their potential for damage on enzymatic activation within lung or liver cells. Since many components of a complex mixture act in concert on these complex enzymatic pathways, some acting synergistically, others antagonistically, still others in a simply additive manner, the final outcome of metabolic action is very difficult to predict. Since concentrations indoors are usually low, however, the interaction effects, which are modeled by a concentration product term, are expected to be negligible, and a linear addition effect model may be sufficient to describe the effects of indoor mixtures of PAH. Damage to macromolecules such as DNA or protein is one consequence, which can result in genetic changes and modulations resulting in cancer. Epidemiologic studies are lacking, and health dangers have been inferred from occupational studies and animal experiments, involving much higher concentrations than those generally encountered indoors.

RADON

Radon (Rn-222) and more specifically, the radioactive elements to which it decays (radon daughters) are of major concern as indoor air pollutants, particularly in residential dwellings. Concern exists not only in the United States but also in several other countries including Canada and Sweden. As a gas, radon is formed in the outdoor air from the decay of radium, which in turn decays from uranium. Both elements are ubiquitous in soil and, hence, radon is always present in varying concentrations in the air. Radon daughters are charged particles when they are formed and they adsorb to particles in the air. Consequently, the principal mode of exposure is through inhalation and the subsequent deposition of the radon daughters in the lung. As emitters of alpha radiation, the daughters are the main source of injury to tissue. Radon has a radiological half-life of 3.8 days while its daughters have a total half-life of only approximately 30 minutes. This relatively short decay time results in the total expenditure of radiation energy within the immediate area of impact, with maximum injury to the local tissue. Such exposure can lead to the high rate of cancer, as has been shown to occur in workers in uranium and other underground mines.

For the general population, the principal exposure to radon is from within the home. Radon readily enters dwellings through cracks and holes in the foundation and subflooring. The presence of radon in homes has not warranted concern until the past few years, perhaps because of the higher air exchange rates common in older structures and because of the lack of monitoring.

The general population receives an estimated annual dose of 200 mrem per year from background radiation. (Radiation workers are limited to an occupational dose of no more than 5000 mrem per year). Approximately half of this amount of 200 mrem (90 mrem) is attributable to exposure to radon (Cothern and Lappenbush 1986). Because the remaining background radiation comes from several sources such as cosmic rays and other naturally occurring radioactive materials, radon is the highest single source of background radiation. Various studies estimate the risk of lung cancer from the natural presence of radon. From these studies the National Council on Radiation Protection and Measurements (NCRP) approximates the average risk as 10×10^{-6} lung cancers per year per person per WLM.*

Major sources of radon in residential dwellings located in areas of the country with soil rich in uranium deposits include the soil underlying and adjacent to these structures, building materials used in the construction of houses, and the water supply to homes. Radon may also be present in natural gas used in the home. However, concentrations of radon from outside and from natural gas are small by comparison and are not considered here.

The emanation of radon from soil is dependent upon the concentration of radium in the soil itself and upon factors that affect its movement through soil, namely, the degree of fractured soil rock and the soil permeability. Pressure driven flow of air from the soil can also occur.

The average concentration of radium (as the source of radon) in the United States is approximately 0.5 to 1.0 pCi per gram of soil, although there is considerable variability. Differences in concentration occur because of two factors: (1) naturally occurring geologic formations in certain parts of the country contain high levels of uranium and (2) manmade areas of high uranium concentrations found in waste tailings from the mining of uranium and phosphate rock. (Phosphate rock contains approximately 0.01% of uranium-238). These manmade sources can have particularly high radium-226 concentrations e.g., 100 to 500 pCi/g in uranium mine tailings (located in the western states) and levels of 6 to 22 pCi/g in areas of Florida that have been reclaimed from earlier phosphate mining activities (NCRP 1984). Problems arise with these sources when, for example, mine tailings are used as fill underneath new homes, homes are built on reclaimed phosphate mining areas, and phosphate slag is used in road building and construction.

Once radon is released from the soil under a structure, its subsequent entry depends on its efficiency in following the path of least resistance into the home, e.g., migration through cracks and holes in the foundation and subflooring and through basement drains (see Figure 2).

Building materials used in house construction can contain radium from which radon is released and enters the structure. Normally the concentrations of these elements are low for materials used in the United States, having a range of 0.003 pCi/l to 0.3 pCi/l (U.S. Department of Energy 1986: Indoor Air Quality Environmental Information Handbook: Radon). Radon from building materials becomes a concern when these levels begin to exceed normal background levels of approximately 1 pCi/g. This is the case in Sweden where, for example, homes built with aerated concrete, consisting of alum shales, contain radium concentrations of 20 to 65 pCi/g. Such homes have radon concentrations four times higher than in those made with other materials (Walsh et al. 1984).

The emanation rate of radon from such materials depends upon the radium concentration in the materials, the porosity and surface finish of the materials, and environmental conditions. Brick, concrete, and wood, however, normally contain insignificant amounts of radium. Average emanation rates for these materials are, for concrete: 0.001 to 0.25 $pCi/m^2 \cdot s$; for bricks and fiberboard gypsum: $<10^{-4}$ $pCi/m^2 \cdot s$; for gypsum board: 0.1 $pCi/m^2 \cdot s$. An increase in moisture content of these materials can cause an increase in emanation rate from 10 to 20% and up to 100%.

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*The working level (WL) is a unit of radon exposure and is a weighted average of the concentration of radon daughters. The working level month (WLM) is the unit of cumulative exposure and is defined as the exposure to 1 WL for 170 hours (an occupational month). Radon exposures of 0.02 WL are suggested maximum levels for inhabited areas, while exposures of 0.05 WL are considered "high" and those of 0.5 WL are "very high." An exposure of 1 WL in homes is equivalent to a concentration of about 200 pCi/L (assuming 50% equilibrium) or a lifetime risk of 1.5×10^{-4} per WLM. Such risk is dependent upon exposure duration and age at first exposure (NCRP 1984).

Tap water can be a significant source of radon in a home when the water source is a deep well. Water having elevated radium levels has correspondingly high radon levels, groundwater being more likely to have higher levels than surface water. As with air, the major concern with water is as a source of inhaled radon rather than ingested radon. The air burden is increased as the gas is released from heated, finely divided droplets of water, as in a shower. Efficiency of gas release is dependent on temperature, surface area exposed to air, and agitation of the water. The efficiency of transfers can vary from practically none for cool, unagitated water, to up to 90% for water agitated and near boiling, as in a dishwasher. Most deep water supplies in the United States have radon concentrations of less than 2000 pCi/L. Some areas, however, such as Maine and Finland, have levels exceeding 100,000 pCi/L (Walsh et al. 1984). The contribution of drinking water to the annual background radiation dose equivalent is 0.2 to 5 mrem per year, approximately 2.5% of the total background (Cothern and Lappenbush 1986).

The average radon level for almost 1400 homes throughout the United States was measured at 1.1 pCi/L. Table 4 shows the results of these measurements and their distribution across the country. As would be expected, higher levels are found in the basements of homes with the concentrations decreasing at higher floor levels. In one study of 169 homes, the average measurements were basement -- 3.87 pCi/L; 1st floor -- 2.19 pCi/L; 2nd floor -- 1.62 pCi/L (Stranden 1980).

The primary route of exposure is via inhalation of radon daughters with their subsequent deposition deep in the lungs. Some daughters may be deposited elsewhere in the body from the decay of radon once it has been inhaled and transported (as a gas) via the bloodstream. However, this transport involves a very small amount of the total daughters, because most of the radon is exhaled, leaving little time for decay to occur.

Approximately 90% of the radon daughters may attach initially to airborne particles, which are then inhaled. Depending on the size of the particles, these daughters may collide with walls of the air passages before reaching the lung. The unattached fraction of radon daughters, the remaining 10%, represents a significant source of exposure, because this fraction has a much higher rate of deposition than the attached fraction and is more efficient in delivering a dose to the critical cells of the lung (Walsh et al. 1984; Stranden 1980).

Overall, 30% of inhaled radon daughters are deposited in the lung, depending upon particle size (for attached daughters) and breathing patterns. The radiological half-life for radon daughters is relatively short (approximately 30 minutes) while the biological half-life is too long to be a factor. Consequently, the daughters will not migrate far from their original site of deposition and will release their energy within a comparatively small volume of tissue.

The major health consequence of a significant exposure to radon is lung cancer. Radiation is known to produce other types of cancer and other types of illness. However, with radon exposure, the total dose is delivered almost exclusively to the lung and even to a particular area of the lung. The alpha radiation emitted by radon daughters expends its relatively high energy in a small volume, thus increasing the probability of significant damage to the surrounding tissue.

Upon inhalation, radon daughters are either deposited along the air passages (primarily "attached daughters") or are carried to the bronchioles and alveoli. This area, particularly the cells just below the surface, receive the maximum dose from radon exposure. Stem cells located in this area divide rapidly and are especially susceptible to radiation damage. If the total dose is sufficiently great, then probability for the development of lung cancer is high.

This observation is borne out by numerous epidemiological studies of lung cancer in underground miners, particularly uranium miners, both in the United States and in other countries. Despite the presence of other possible carcinogens, such as uranium ore dust, silica dust, and diesel fuel exhaust fumes, an association has been established between the incidence of lung cancer in these miners and their accumulated exposure to radon daughters. It is difficult, however, to extrapolate the results from the mining experience to the general population. Not only are there deficiencies in those studies, but the general population differs considerably from the mining population. The general population has different spatial, temporal, age, and sensitivity distributions and is exposed to complex mixtures of which radon is but one component.

These studies reveal several factors that are involved in the relationship between radon exposure and the resultant lung cancer in man:

1. The latent period for lung cancer varies inversely with age at first exposure, and with cigarette smoking, and with total exposure and/or exposure rate. Thus, the shortest latent periods are found among those men who are elderly at the start of mining, who smoke heavily, and who have the most intense exposures. The latent period has a range of 7 to 50 years with mean values between 20 and 30 years. No significant appearance of lung cancer has appeared before age 40. Thus, the concept of a fixed latent period is not supported since radon daughter-induced cancer tends to appear at ages normally anticipated, although many of the cancers do appear between ages 40 and 55 among miners who smoke.

2. The incidence of lung cancer in exposed miners is largely dependent on dose, but there are other factors involved. Incidence appears to vary directly with age at first exposure, with number of cigarettes smoked, with age of individuals at time of observation, and with length of observation period.

3. Results from the studies are not suitable for determining whether a threshold exposure for lung cancer induction exists. This finding is in keeping with the current view of radiation biology and radiation protection in that radiation-induced cancer is a stochastic process, one for which the probability of an effect occurring, rather than its severity, is a function of dose and is without a threshold, below which no effects occur (DOE 1986).

In summary, none of these occupational studies show a statistically significant excess of lung cancer for cumulative exposure <60 WLM. Therefore, to estimate the effect of radon daughter exposure at environmental levels (normally, <20 WLM per lifetime), the attributable risk at low exposures must be extrapolated from that at high exposures.

Lifetime lung cancer risks have been developed from these epidemiology studies. The NCRP has calculated these risks for radon exposure in mines and under environmental conditions which are summarized in Table 5. Additional risk projections have been estimated by source of calculations and are displayed in Table 6. The EPA risk estimate is based on a 2% risk coefficient. Lifetime risk may vary by a factor of ±2. All of the risk estimates listed are based on the same few epidemiologic studies involving uranium miners. Populations exposed to indoor radon differ from the worker population in their susceptibility to radiation damage. Infants and children, because of their rapidly growing cells, are more sensitive to radiation injury, and elderly people may have reduced defense mechanisms. Smoking history, which influences the probability of radiation-induced cancers, also tends to differ in at-home populations compared to those at work. Another consideration is that exposure times may be much longer for at-home populations. The working level (WL) is based on an exposure time of a 170-hour occupational month. At-home exposure for some individuals can be 24 hours per day or 720 hours per month.

VOLATILE ORGANIC CHEMICALS (VOC)

In addition to combustion products and other pollutants found in indoor environments, the class of contaminants known as "volatile organic compounds" (VOC) poses a number of health effect possibilities. VOC are found in homes as products of emissions from building materials (floor coverings, furnishings, paint), consumer products (polishes, cleansers, solvents), combustion (from cigarettes, kerosene heater, wood stove), and other miscellaneous categories (cooking, ambient air intrusion). In 1978, Mølhave and Moller reported that the VOC in Danish buildings were higher than the VOC in the ambient air, indicating the importance of indoor VOC sources and exposures. Later work by Wallace (1984), Jarke (1979), Lebret et al. (1984), DeBortoli et al. (1984) and Mage and Gammage (1985) have confirmed this relationship in residences in the U.S., Italy, and Germany. Total concentrations of VOC in new homes (while building materials are outgassing) have been reported up to 19 mg/m^3, and these concentrations are reduced by an order of magnitude in older occupied homes. These concentrations may be of concern to human health with respect to acute and chronic health effects. Mølhave (1984) has demonstrated acute neurological effects from a mixture of 22 VOC found in Danish buildings at levels of 5 mg/m^3 to 25 mg/m^3, and he has hypothesized that the total mixture is acting on the trigeminal nerves to produce sensorial and behavioral effects.

Volatile organic compounds have certain characteristics in common. All are easily vaporized and dissolve readily in lipids, and, therefore, are transported across cell membranes and are absorbed by the lung, skin, and gastrointestinal tract of animals and humans. Many VOC are irritants, and/or are neurotoxic agents, and/or can act as carcinogens, co-carcinogens, or promoters.

Up to 300 volatile organic compounds have been detected in homes during the TEAM (Total Exposure Assessment Methodology) study conducted by EPA. Between 10 and 20 of these may exist in concentrations that could be of concern. Of these, benzene, carbon tetrachloride, chloroform, methylene chloride, dinitrotoluene, nitrosamines, and phenols, among others, have been assessed for lifetime cancer risk by the EPA Carcinogen Assessment Group. The possible effects of chronic lifetime exposures to the individual compounds may be expressed as an increased probability of contracting cancer due to the carcinogenic nature of some of these compounds. For example, Wallace measured an average of 10 $\mu g/m^3$ of chloroform in Elizabeth, NJ, homes. The EPA/CAG estimated the 95% upper bound risk as 7×10^{-2} per mg chloroform/kg body weight-day (if 100 people have an uptake of 1 mg per kg per day over their lifetime, then seven excess cancers would be expected in that population).

1,1,1-Trichloroethane (methyl chloroform). Methyl chloroform is a volatile chlorinated hydrocarbon that is widely used as a solvent in consumer products such as spot removers and other cleaning products. It has been estimated that 88% of the annual production of methyl chloroform (320,000 metric tons) is released to the atmosphere through dispersive use. This is significant when considering the compound's widespread indoor application. The concentration/human response relationships are shown in Table 7. Maximum indoor concentrations of methyl chloroform in residences have been measured or estimated to be in the range of 10 to 50 ppm.

Few animal studies have been done on nervous system effects and behavior. No studies for teratogenic, carcinogenic, or mutagenic effects have been completed. Such research is needed to determine health effects of this compound.

Styrene (vinyl benzene). Styrene is used in the manufacture of plastics and synthetic rubber products. Styrene may be released to indoor environments through the thermal breakdown of its polymers. Styrene is rapidly absorbed by the gastrointestinal tract or through the skin. Also, when taken up by inhalation, styrene is absorbed proportional to its atmospheric concentration.

Styrene's main effect on humans centers around irritation to the skin, eyes, and respiratory tract. Styrene also causes neurotoxic and behavioral effects. Some reported effects and concentrations are presented in Table 8.

Strong epidemiologic studies concerning low-level, chronic exposure to styrene are lacking. Workplace exposure studies indicate some low-level nervous system effects, but these exposure levels are at higher concentrations than people breathing indoor air are generally exposed to.

Toluene. Toluene is the most prevalent hydrocarbon in the atmosphere. Levels generally range in outside air from 0.14 to 57 ppb. Three million metric tons are produced annually. Persons may be exposed to toluene through inhalation of air, ingestion of food or water, or dermal exposure. Some common sources of exposure to toluene include gasoline vapors, paint and varnish thinners and removers, and solvents for adhesives, inks, and pharmaceuticals. The known concentration-effect relationship of toluene is depicted in Table 9.

Current health information for toluene is limited and suggests that toluene may not be a serious hazard at the concentration measured in indoor environments. However, additional investigation is required especially to better assess day-to-day residential and office exposures. In regard to neurobehavioral toxicity and carcinogenicity, the general population's exposure to toluene indoors remains unevaluated. Data are needed on the magnitude, frequency, duration, and extent of toluene exposure from indoor sources. Also, it is clear that the central nervous system (CNS) is the most sensitive physiological system responding to toluene. CNS data of short-term high-level exposure to toluene are available. However, the CNS response to long-term exposure at lower toluene concentrations is not now available. The role of toluene in cancer is currently expected as being a weak one. However, long-term animal studies are necessary to verify this position.

It is not possible at the present time to assess the variety of individual effects associated with the hundreds of VOCs found in indoor environments. The problem faced is one of determining what specific organic pollutant should be singled out for closer scrutiny.

For the purpose of prudent public health protection, it is suggested that pollutants of immediate concern and in need of assessment for indoor environments be evaluated in terms of the following characteristics:

1. They are known or suspected carcinogens to either humans or animals and/or they are known or suspected neurotoxicants to humans as determined from animal studies or epidemiology studies.

2. They are present in U.S. residences as determined from results of previous monitoring studies or they are expected to be present from knowledge of emissions from common sources such as tobacco smoking, solvents, woodstove emissions, or a variety of consumer products.

3. They are identifiable and quantifiable using reliable analytical techniques.

Chemicals that have these characteristics and which have been previously found in U.S. residences are listed below.

vinyl chloride
methylene chloride
chloroform
carbon tetrachloride
1,1,1-trichloroethane
trichloroethylene
tetrachloroethylene
benzene
o-xylene
m,p-xylene
m,p-dichlorobenzene
ethylbenzene
styrene
1,2-dichloroethane
1,1,1,2-tetrachloroethane
1,1,2,2-tetrachloroethane
1,1,1-trichloroethane
1,1,2-trichloroethanedecane
hexachloroethane
decane
chlorobenzene
ethylphenol
acrolein
toluene
vinylidene chloride

The purpose of listing these compounds is to emphasize the need to examine the chronic and acute effects these substances may pose to individuals who are exposed to them continuously in their homes and workplaces.

Mølhave (1984) has taken a different generic-total-VOC approach to exploring health effects for indoor exposure in a controlled exposure, double blind experiment. When otherwise healthy but sensitive subjects were exposed to a mixture containing the following 22 organic gases and solvent vapors found in Danish homes, they experienced those symptoms associated with the sick or tight building syndrome.

n-Hexane
n-Nonane
n-Undecane
1-Octene
1-Decene
Cyclohexane
3-Xylene
Ethylbenzene
1,2,4-Trimethylbenzene
n-Propylbenzene
α-Pinene
n-Pentanal
n-Hexanal
Iso-propanol
n-Butanol
2-Butanol
3-Methyl-3-Butanone
4-Methyl-2-Pentanone
n-Butylacetate
Ethoxyethylacetate
1,2-Dichlorethane

The symptoms include reduced power of concentration; irritation of eyes, nose, and throat; and general malaise. Mølhave observed a clear health effect for concentrations of the VOC mixture in the range 5 mg/m^3 to 25 mg/m^3. Such indoor concentrations of VOC mixtures are not uncommon, especially in new Danish residences.

Mølhave has hypothesized that some of the sick building syndrome symptoms can be best explained by a total mixture, rather than exposure to an individual compound. He hypothesized that a summation of the 22 VOC in the range 5 mg/m^3 to 25 mg/m^3 contributes to a nonspecific sensorial stimulus of the trigeminal nerve. The removal of any one VOC from the

total mixture is not expected to reduce the overall effect. Further, a mixture of 22 different VOC, perhaps from the U.S. list, above would produce similar effects.

Although Mølhave's findings are plausible and interesting, it is important to realize that it is necessary to first test his hypothesis and then to properly assess the health implications of indoor concentrations of VOC. In summary, variability of human sensitivity to different chemicals is at the heart of the indoor health issue. Human responses may occur as a result of exposure to either single VOC or VOC in total. However, much additional investigation is necessary to determine the relative importance of each type of exposure and response.

The pollutants having an impact on indoor air quality considered in this paper are limited to formaldehyde, some products of combustion, some volatile organic compounds, and radon. More information concerning the human carcinogenic potential of formaldehyde is needed. While the effects of carbon monoxide are clear at high concentrations, more information concerning health effects at low levels, especially among physically compromised individuals, is required. Especially lacking is clear information regarding the long-term effects of particulates and the polynuclear aromatic hydrocarbons (PAH) that comprise part of the complex mixtures of combustion products. Also lacking is information on the prevalence, composition, and effects of exposure to mixtures of volatile organic compounds found in homes. This paper does not explore categories of indoor pollutants such as fibers, biological contaminants, pesticides, or dyes, which represent classes of pollutants whose roles in the total mixture of indoor air also need consideration.

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TABLE 1
Effects of Formaldehyde in Humans After Short-term HCHO Respiratory Exposure (concentrations in mg/m^3; 1 mg/m^3 = 0.813 ppm)

Reported Ranges	Estimated Median	Effect
0.06-1.2	0.1	Odor threshold 50% of people
0.01-1.9	0.5	Eye irritation threshold
0.1-3.1	0.6	Throat irritation threshold
2.5-3.7	3.1	Biting sensation in nose, eye
5-6.7	5.6	Tearing eyes, long term lung effects
12-25	17.8	Tolerable for 30 minutes with strong flow of tears lasting 1 hour
37-60	37.5	Inflammation of lung (pneumonitis), edema, respiratory distress: danger to life
60-125	--	Death

Source: National Center for Toxicological Research (1984).

TABLE 2
Health Effects of Carbon Monoxide (Explains Figure 1)

Range Category, Figure 1	% COHb	Effects
A	0.3-0.7	Normal physiologic norm for non-smokers
B	0.7-2.9	No demonstrated changes in functions
C	2.9-4.5	Affects cardiovascular system of compromised patients (decreased exercise performance in patients with angina; increased duration of angina attacks)
D	4-6	Behavior affected (decreased vigilance; reduced maximal work capacity; lassitude; norm for smokers)
E	7-10	Cardiovascular effects in healthy adults (P-wave EKG changes)
F	10-20	Slight headaches; lassitude; breathlessness from exertion; potential damage to fetuses
G	20-30	Severe headaches, nausea, abnormal manual dexterity
H	30-40	Severe headaches; irritability; impaired judgment; dimmed vision; nausea and vomiting; weak muscles
I	40-50	Fainting; convulsions; coma
J	60-70	Coma; depressed cardiac activity and depressed respiration; usually fatal

Source: U.S. Environmental Protection Agency (1979); U.S. Environmental Protection Agency (1984).

TABLE 3
Human Response to Short-term NO_2 (alone) Exposure
(1 mg/m^3 NO_2 = 0.532 ppm)

NO_2 Concentration, mg/m^3	Exposure time	Effect
0.23	Not reported	Odor threshold
0.14-0.50	Not reported	Threshold for dark adaptation
0.5-5.0	15 minutes	Increased airway resistance in asthmatics
7.5-9.4	15 minutes	Decreases pulmonary diffusing capacity
9.4	5 minutes	Alveolar-arterial pO_2 difference
11.3-15.2	5 minutes	Increased airway resistance in asthmatics and healthy adults

Source: U.S. Department of Energy (1986).

TABLE 4
Radon-222 Concentrations Measured in U.S. Residences

Location	No. of Houses	Randon-222 Concentration GM(pCi/l)	GSD
Washington	138	0.71	2.48
Oregon	90	0.86	2.27
San Francisco, CA	29	0.35	2.19
Las Vegas, NV	3	0.79	2.47
Montana	20	1.32	2.34
Butte, MT	179	3.46	3.03
Fargo, ND	11	5.69	2.39
Idaho	17	1.30	2.94
Colorado Springs, CO	16	1.87	2.34
Grand Junction, CO	62	1.39	1.61
New Mexico	9	2.31	2.45
Santa Fe, NM	6	0.94	2.26
Farmington, NM	6	0.74	1.66
Houston, TX	103	0.47	1.94
Wisconsin	50	1.10	2.17
Chicago, IL	44	0.50	3.00
Portland, ME	11	0.44	2.98
Central Maine	70	1.66	2.56
New York State	27	0.89	2.65
Lewiston, NY	10	0.50	1.31
Rochester, NY	8	0.50	4.08
Northeastern NY	9	0.78	3.12
New York City area	11	0.45	1.56
New Jersey	9	1.36	1.73
Princeton, NJ	11	1.09	3.95
Middlesex, NJ	15	0.52	1.39
Pittsburgh, PA	122	1.45	2.35
Philadelphia, PA area	31	1.84	2.32
Cannonsburg, PA	8	0.94	2.15
Eastern Pennsylvania	36	3.29	3.41
Maryland	58	1.90	3.60
Damacus, MD	41	3.04	2.11
East Tennessee	40	2.00	2.50
Oak Ridge, TN	14	1.28	2.24
Raleigh, NC	10	0.52	2.80
Charleston, SC	20	0.47	2.12
Florida (non-mined)	29	0.73	1.60
Florida (mined)	4	2.14	2.51

Source: Nero et al. (1984).

TABLE 5
Lifetime Lung Cancer Risk From Radon Exposure in Mines and Under Environmental Conditions

Exposure Duration	Lung Cancers in a Population of 10^5 Persons: Risk Per Rad of Radon Exposure in Mines	Risk per WLM per Year Under Environmental Conditions	Risk per pCi $^{222}Rn/m^3$ Under Environmental Conditions
1 year	19	13	0.0051
5 years	94	66	0.026
10 years	190	130	0.051
30 years	540	380	0.14
Life	800	560	0.21

Source: NCRP (1984).

TABLE 6
Summary of Lifetime Cancer Risk Estimates from Radon Daughter Exposure

Source	Lifetime Risk of Lung Cancer (10^{-4}/WLM)
NCRP #78 (1984)	1 - 2
ICRP (1981)	1.5 - 4.5
Evans (1981)	1
UNSCEAR (1982)	2 - 4.5
NAS(BEIR) (1980)	7.3
EPA (1985)	7.6
AECB (1982)	6.0

NCRP - National Council on Radiation Protection and Measurements
ICRP - International Commission on Radiological Protection
UNSCEAR - United Nations Scientific Committee on the Effects of Atomic Radiation
NAS(BEIR) - National Academy of Sciences (Effects on Populations of Exposure to Low Levels of Ionizing Radiation: 1980)
EPA - Environmental Protection Agency
AECB - Atomic Energy Control Board - Canada

TABLE 7
Dose Effect Relationships of 1,1,1-Trichloroethane

Concentration	Methyl Chloroform Effects
100 ppm (540 mg/m^3)	Apparent odor threshold
350-500 ppm (1,890-2,700 mg/m^3)	Obvious odor, slight changes in perception
1,000 ppm (5,400 mg/m^3)	Disturbances of equilibrium
1,900-2,650 (10,260-14,310 mg/m^3)	Lightheadedness, irritation of the throat
> 5,000 ppm (27,000 mg/m^3)	Onset of narcosis

Source: U.S. Environmental Protection Agency (1985).

TABLE 8
Dose-Effect Relationships of Styrene

25-500 ppm (106-2125 mg/m^3)	Reduced reaction times
100 ppm (425 mg/m^3)	Behavioral effects - fatigue, headache, poor memory
200 ppm (900 mg/m^3)	Eye and respiratory tract irritation
800 ppm (3400 mg/m^3)	Immediate eye and throat irritation Loss of balance

Source: U.S. Environmental Protection Agency (1986).

TABLE 9
Dose-Effect Relationships of Toluene

> 37 ppm (~150 mg/m^3)	Probably perceptible to most humans.
50 to 100 ppm (~200 to 400 mg/m^3)	Subjective complaints (fatigue or headache) but probably no observable impairment of reaction time or coordination.
200 ppm (~750 mg/m^3)	Mild throat and eye irritation.
100 to 300 ppm (~400 to 1100 mg/m^3)	Detectable signs of incoordination may be expected during exposure periods up to eight hours.
400 ppm (~1500 mg/m^3)	Lacrimation and irritation to the eyes and throat.
300 to 800 ppm (~1100 to 3000 mg/m^3)	Gross signs of incoordination may be expected during exposure periods up to eight hours.

Source: U.S. Environmental Protection Agency (1983).

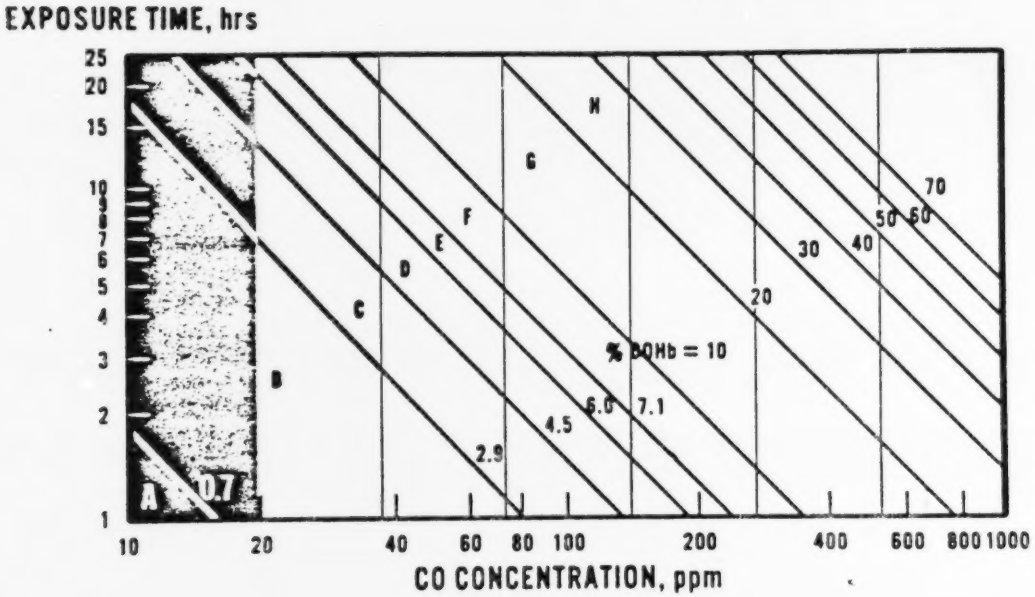

Figure 1. Percent COHb resulting from exposure time relative to exposure concentration

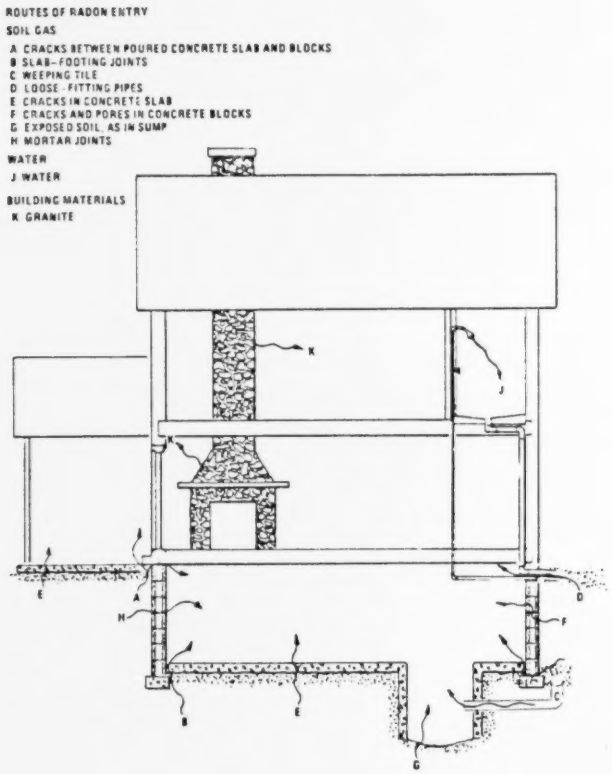

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Figure 2. Major pathways for entry of radon into houses